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EXAMINER	
SITTON, JEHANNE SOUAYA	
ART UNIT	**PAPER NUMBER**
1634	

DATE MAILED: 07/06/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/608,296	ADAM ET AL.	
	Examiner	Art Unit	
	Jehanne S. Sitton	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *18 April 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-15* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-15* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *27 June 2003* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date *11/03, 11/05*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Election/Restrictions

1. Applicant's election without traverse of Group I, claim 3-4, 8, and 12-13 in the reply filed on 4/10/2006 is acknowledged. Applicants further election of SEQ ID NO: 38 for claim 8 in the reply filed on 4/18/2006 is acknowledged. Because applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election of a specific SEQ ID NO: has been treated as an election without traverse (MPEP § 818.03(a)).

2. Upon examination of the application, it was found that searching positions 9182 and 7328 of SEQ ID NO: 1 was not burdensome. The examiner has additionally rejoined SEQ ID NO: 40. Accordingly, the restriction requirement between the positions (groups I and II) and SEQ ID NOS 38 and 40 is withdrawn and an action on the merits of claims 1-15, SEQ ID NOS 38 and 40 is set forth below. Additionally, it is noted that the restriction requirement between SEQ ID NOS 38, 40, 61 and 62 has been amended to be a species requirement as set forth below.

3. Claim 7 is generic to the following disclosed patentably distinct species: SEQ ID NOS 38, 40, 61, and 62. The species are independent or distinct because they are drawn to structurally distinct nucleic acid molecules. Upon the allowance of a generic claim, applicant will be entitled to consideration of claims to additional species which depend from or otherwise require all the limitations of an allowable generic claim as provided by 37 CFR 1.141. MPEP § 809.02(a).

Specification

4. The disclosure is objected to because it contains an embedded hyperlink and/or other form of browser-executable code. Applicant is required to delete the embedded hyperlink and/or other form of browser-executable code. See MPEP § 608.01.

Claim Objections

5. Claims 4, 6, 13, and 15 are objected to under 37 CFR 1.75(c), as being of improper dependent form for failing to further limit the subject matter of a previous claim. Applicant is required to cancel the claim(s), or amend the claim(s) to place the claim(s) in proper dependent form. The claims are dependent from claims 3, 5, 12, and 14, respectively, which each set forth a particular polymorphic position. However, claims 4, 6, 13, and 15 are limited to a different polymorphic position and therefor do not further limit the scope of the claims from which they directly depend, but set forth alternative embodiments. Accordingly, in meeting the limitation of claim 4, for example, which is drawn to detection of a single variation, one could not perform the method of claim 3, which is drawn to detection of a different variation. Claim 4 is drawn to an alternative step, rather than an additional step and fails to limit the claim from which it directly depends.

Claim Rejections - 35 USC § 112

6. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

7. Claims 1-15 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of diagnosing leanness in an a human subject comprising detecting the presence of an A at position of 7328 of SEQ ID NO: 1 or a G at position 9182 of SEQ ID NO: 1, wherein the presence of an A at position 7328 or a G at position 9182 of SEQ ID NO: 1 is indicative of a predisposition to leanness, does not reasonably provide enablement for a method of diagnosing predisposition to increased fat deposition or leanness in a subject by detecting any polymorphic variation in the nucleotide sequence of SEQ ID NO: 1, a nucleotide which encodes the polypeptide of SEQ ID NO: 2, a nucleotide sequence which encodes a polypeptide that is 90% identical to SEQ ID NO: 2, a fragment of any such nucleotide sequence, or a method of diagnosing increased fat deposition or leanness by detecting a G at position 7328 of SEQ ID NO: 1, or a method of diagnosing predisposition to increased fat deposition or leanness in a subject by detecting any polymorphic variation in linkage disequilibrium with the guanine at position 7328 of SEQ ID NO: 1. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims. There are many factors to be considered when determining whether there is sufficient evidence to support determination that a disclosure does not satisfy the enablement requirements and whether any necessary experimentation is undue. These factors have been described by the court in *In re Wands*, 8 USPQ2d 1400 (CA FC 1988). *Wands* states at page 1404,

> "Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of

those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

The nature of the invention and the breadth of the claims:

The claims encompass detecting any type of fat deposition (increased or decreased – [leanness] relative to average fat deposition in a population) in any subject by detecting any polymorphic variation in a the nucleotide sequence of SEQ ID NO: 1, a nucleotide which encodes the polypeptide of SEQ ID NO: 2, a nucleotide sequence which encodes a polypeptide that is 90% identical to SEQ ID NO: 2, or a fragment of any such nucleotide sequence. The claims further encompass a method of diagnosing increased fat deposition or leanness in any subject by detecting a G at position 7328 or T at position 9182 of SEQ ID NO: 1. The claims also encompass a method of diagnosing predisposition to increased fat deposition or leanness in any subject by detecting any polymorphic variation in linkage disequilibrium with the guanine at position 7328 or thymine at position 9182 of SEQ ID NO: 1.

The nature of the claimed invention, therefore, requires the knowledge of predictive associations between any polymorphism in any of the recited nucleic acids, or any polymorphism in linkage disequilibrium with such, in any subject and a predisposition to fat deposition.

The amount of direction or guidance and presence/absence of working examples:

The specification teaches that SEQ ID NO: 1 is the PLA2G1B nucleotide sequence. The specification teaches comparison of sequences from human PLA2G1B (SEQ ID NO: 1) and the PLA2G1B sequence from rat, mouse, and sand rat (figure 5A). The specification teaches that individuals were tested for central fat measurement and triglyceride measurements (page 41).

The specification teaches that individuals in the top and lower 10th percentile were chosen as subjects and that a subset of individuals falling in the middle range were chosen as a control group. The specification teaches that potential polymorphisms in the PLA2G1B polynucleotide were identified in a publicly available SNP database and verified in a group other than the study group (page 44). The specification teaches 10 SNPs (page 44, table 1, page 49) were found to be statistically significant polymorphisms. However, the specification teaches that only two of these SNPs, at positions 7328 and 9182 of SEQ ID NO: 1 were found to have a statistically significant association with reduced fat deposition (leanness) (A: p=0.00669; G: p=0.00688, respectively). The specification does not teach if the alternate allele was significantly associated with increased fat deposition or whether it is prevalent in the population in people with average fat deposition.

Although the specification teaches that the SNPs at positions 7328 and 9182 are in strong linkage disequilibrium (page 49, table 7), the specification does not teach any other polymorphisms in linkage disequilibrium with position 7328 or 9182 of SEQ ID NO: 1. It is clear from table 7, that a polymorphism, simply by virtue of being found in SEQ ID NO: 1, is not necessarily linked to the SNP at position 7328 or 9182. The specification provides no predictable correlation as to the identity of any other alleles in linkage disequilibrium with position 7328 or 9182.

The claims encompass not only detection of any polymorphism in SEQ ID NO: 1, but in sequences which encode SEQ ID NO: 2, sequences which encode a polypeptide with 90% identity to SEQ ID NO: 2, as well as sequences comprising fragments of such. The claims therefore encompass detection of polymorphisms in a large genus of homologs of SEQ ID NO:

1, from any source. However, the specification does not teach degenerate variants of SEQ ID NO:1, nor does the specification teach any homologs of SEQ ID NO: 1 which encode a polypeptide with 90% identity with SEQ ID NO: 2. Although the specification teaches PLA2G1B in mouse, rat, and sand rat, it does not teach the sequence from any other mammalian species, such as dog, cat, monkey, etc. The specification does not teach any polymorphisms whatsoever, in any of the sequences encompassed by sections b-d of claims 1 and 11, or any polymorphisms in any PLA2G1B sequences in any other species.

The specification provides no universal correlation that any SNP in any of the claimed nucleic acids would be associated with increased or decreased fat deposition. Of 10 disclosed SNPs, the specification teaches an association between only 2 SNPs and reduced fat deposition, thus it is clear that "any" polymorphism in the encompassed nucleic acids would not be predictable associated with increased or decreased fat deposition in any subject. Additionally, the specification provides no guidance as to how the SNPs at position 7328 (A) and 9182 (G) function to provide a phenotype of reduced fat deposition. The specification provides no structure/function correlation between the disclosed SNPs and reduced fat deposition for the ordinary artisan to be able to predict which other positions within the claimed sequences might be predictably associated with the claimed phenotypes. It is not known whether this position exists in other variants or homologs or other mammalian genes or what a "corresponding" position would be in another gene or whether a polymorphism would have the same effect in another gene, or what the identity of that polymorphism might be. Therefore, the skilled artisan would be unable to predictably correlate any other structural change in any other region of PLA2G1B in any other species. The two alleles: an A at position 7328 and a G at position 9128,

could be part of a reduced fat deposition-associated haplotype, however the causative mutation is not necessarily one of the SNPs taught in the specification. The causative mutation could be in a gene thousands of nucleotides away, however the specification provides no indication of what this allele might by.

The specification provides no predictable association that any alteration, in any PLA2G1B gene, in any subject, is diagnostic for increased or decreased fat deposition. No common element or attributes of the sequences are disclosed which would permit selection of sequences as phenotypically associated polymorphisms. No structural limitations or requirements which provide guidance on the identification of sequences which meet these functional limitations of associating a polymorphism with fat deposition is provided. Further, these claims expressly encompass allelic variants including insertions, deletions, substitutions and transversions at thousands of different sites. No written description of alleles, of upstream or downstream regions containing additional sequence, which are associated with any phenotype are described in the specification. Additionally, the specification provides no evidence that any SNP at such position, in either humans, or mice or dogs for example, provides a predictable association with fat deposition. The polymorphisms shown are not representative of the genus of any polymorphism associated with fat deposition because it is not clear which polymorphisms within "any" PLA2G1B gene would have the same affect. It is not clear whether the polymorphisms shown are causative for the detected phenotype or whether they may simply represent markers for another gene that is in linkage disequilibrium with the specific alleles at issue, and the actual gene which is involved in the detected reduced fat deposition may be tens of thousands of nucleotides distant from the polymorphisms described in the specification. The

specification does not teach the function of polymorphisms of PLA2G1B nor how their function, or lack of function, or altered function are predictably associated with fat deposition.

The state of the prior art and the predictability or unpredictability of the art:

The art does not teach the function of polymorphisms of PLA2G1B or how they are involved in fat deposition, either in humans or in non human species.

The level of skill in the art:

The level of skill in the art is deemed to be high.

The quantity of experimentation necessary:

The quantity of experimentation in this area is extremely large as it requires analysis of each position in "any" PLA2G1B gene to determine whether any alteration at each position is associated with any increased or reduced fat deposition. As neither the art nor the specification provide guidance as to which alterations at positions throughout PLA2G1B are predictably associated with fat deposition, such analysis is replete with trial and error experimentation, with the outcome of each analysis being unpredictable. Screening each possible alteration in any PLA2G1B gene represents an inventive and unpredictable undertaking in itself, with each of the many intervening steps, not providing any guarantee of success.

In order to practice the invention as claimed, one would first have to establish that a predictive relationship exists between the disclosed polymorphisms and increased fat deposition in any subject. Further, the scope of many of the claims requires knowledge of an association

between all mutations in any PLA2G1B gene and reduced or increased fat deposition in humans or any mammalian species. Due to the scope of the claims, one of skill in the art would be required to further undertake extensive trial and error experimentation with a large number of patients with different degrees of fat deposition, and controls, to determine mutations that share a predictive predisposition to increased or reduced fat deposition.

Thus, given the broad claims in an art whose nature is identified as unpredictable, the state of the prior art, the lack of guidance in the specification, the breadth of the claims and the quantity of experimentation necessary to practice the claimed invention, it would require undue experimentation to practice the invention commensurate in scope with the claims.

8. Claims 1-15 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The claims are drawn to detecting any type of fat deposition (increased or decreased [leanness] relative to average fat deposition in a population) in any subject by detecting any polymorphic variation in a the nucleotide sequence of SEQ ID NO: 1, a nucleotide which encodes the polypeptide of SEQ ID NO: 2, a nucleotide sequence which encodes a polypeptide that is 90% identical to SEQ ID NO: 2, or a fragment of any such nucleotide sequence. The claims further encompass a method of diagnosing increased fat deposition or leanness in any subject by detecting a G at position 7328 or a T at position 9182 of SEQ ID NO: 1. The claims

also encompass a method of diagnosing predisposition to increased fat deposition or leanness in any subject by detecting any polymorphic variation in linkage disequilibrium with the guanine at position 7328 or thymine at position 9182 of SEQ ID NO: 1.

The claims encompass not only detection of any polymorphism in SEQ ID NO: 1, but in sequences which encode SEQ ID NO: 2, sequences which encode a polypeptide with 90% identity to SEQ ID NO: 2, as well as sequences comprising fragments of such. The claims therefore encompass detection of polymorphisms in a large genus of variants and homologs of SEQ ID NO: 1, from any source. However, the specification does not teach degenerate variants of SEQ ID NO:1, nor does the specification teach any homologs of SEQ ID NO: 1 which encode a polypeptide with 90% identity with SEQ ID NO: 2. Although the specification teaches PLA2G1B in mouse, rat, and sand rat, it does not teach the sequence from any other mammalian species, such as dog, cat, monkey, etc. The specification does not teach any polymorphisms whatsoever, in any of the sequences encompassed by sections b-d of claims 1 and 11, or any polymorphisms in any PLA2G1B sequences in any other species.

The current claims encompass a large genus of nucleic acids which comprise polymorphisms in any region of any PLA2G1B gene or homolog from any source. The genus includes an enormous number of polymorphisms and mutations for which no written description is provided in the specification. This large genus is represented in the specification by only the particularly named 2 polymorphisms for which data is provided. Thus, applicant has express possession of only 2 particular polymorphisms in SEQ ID NO: 1 which are associated with leanness, in a genus which comprises hundreds of millions of different possibilities. Here, no common element or attributes of the sequences are disclosed which would permit selection of

sequences as polymorphisms. No structural limitations or requirements which provide guidance on the identification of sequences which meet these functional limitations of associating a polymorphism with increased or decreased fat deposition is provided. Further, these claims expressly encompass allelic variants including insertions, deletion, substitutions and transversions at thousands of different sites. However, no predictable correlation between the structural alterations of the 2 polymorphisms disclosed and leanness is provided by the specification. The specification does not teach the function of polymorphisms of PLA2G1B nor how their function, or lack of function, or altered function are predictably associated with fat deposition.

The specification teaches that potential polymorphisms in the PLA2G1B polynucleotide were identified in a publicly available SNP database and verified in a group other than the study group (page 44). The specification teaches 10 SNPs (page 44, table 1, page 49) were found to be statistically significant polymorphisms. However, the specification teaches that only two of these SNPs, at positions 7328 and 9182 of SEQ ID NO: 1 were found to have a statistically significant association with reduced fat deposition (leanness) (A: p=0.00669; G: p=0.00688, respectively). The specification does not teach if the alternate allele was significantly associated with increased fat deposition or whether it is prevalent in the population in people with average fat deposition.

The claims also encompass polymorphisms in linkage disequilibrium with the SNP at position 7328 or 9182 of SEQ ID NO: 1. Although the specification teaches that the SNPs at positions 7328 and 9182 are in strong linkage disequilibrium (page 49, table 7), the specification does not teach any other polymorphisms in linkage disequilibrium with position 7328 or 9182 of

SEQ ID NO: 1. It is clear from table 7, that a polymorphism, simply by virtue of being found in SEQ ID NO: 1, is not necessarily linked to the SNP at position 7328 at 9182.

The specification provides no guidance that any alteration, in any PLA2G1B gene, in any subject, is diagnostic for increased or decreased fat deposition. No common element or attributes of the sequences are disclosed which would permit selection of sequences as phenotypically associated polymorphisms. No structural limitations or requirements which provide guidance on the identification of sequences which meet these functional limitations of associating a polymorphism with fat deposition is provided. Further, these claims expressly encompass allelic variants including insertions, deletions, substitutions and transversions at thousands of different sites. No written description of alleles, of upstream or downstream regions containing additional sequence, which are associated with any phenotype are described in the specification. Additionally, the specification provides no evidence that any SNP at such position, in either humans, or mice or dogs for example, provides a predictable association with fat deposition. The polymorphisms shown are not representative of the genus of any polymorphism associated with fat deposition because it is not clear which polymorphisms within "any" PLA2G1B gene would have the same affect. It is not clear whether the polymorphisms shown are causative for the detected phenotype or whether they may simply represent markers for another gene that is in linkage disequilibrium with the specific alleles at issue, and the actual gene which is involved in the detected reduced fat deposition may be tens of thousands of nucleotides distant from the polymorphisms described in the specification. The specification provides no guidance that the specific alleles exist in other species, therefore, there is no teaching or guidance as to the identity of alleles in linkage disequilibrium with recited alleles in other

species. The specification fails to provide any teaching or guidance as to what the structure of phenotypically associated alleles would be in variants or homologs of SEQ ID NO: 1 in humans, or in PLA2G1B or variants or homologs in other species.

In analysis of the claims for compliance with the written description requirement of 35 U.S.C. 112, first paragraph, the written description guidelines note regarding genus/species situations that "Satisfactory disclosure of a ``representative number" depends on whether one of skill in the art would recognize that the applicant was in possession of the necessary common attributes or features of the elements possessed by the members of the genus in view of the species disclosed." (See: Federal Register: December 21, 1999 (Volume 64, Number 244), revised guidelines for written description.) In the instant case, the specification fails to teach the necessary common attributes or features of the genus of encompassed nucleic acids and polymorphisms in view of the species disclosed. As such, one of skill in the art would not recognize that applicant was in possession of the genus of nucleic acids and polymorphisms encompassed by the broadly claimed invention.

Vas-Cath Inc. v. Mahurkar, 19 USPQ2d 1111, makes clear that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of the invention. The invention is, for purposes of the 'written description' inquiry, whatever is now claimed." (See page 1117.) The specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is claimed." (See Vas-Cath at page 1116.)

The skilled artisan cannot envision the detailed chemical structure of the encompassed nucleic acids and polymorphisms, regardless of the complexity or simplicity of the method of

isolation. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it. The nucleic acid itself is required. See Fiers v. Revel, 25 USPQ2d 1601, 1606 (CAFC 1993), and Amgen Inc. V. Chugai Pharmaceutical Co. Ltd., 18 USPQ2d 1016. The current situation is a definition of the compound solely based on its functional utility, as a polymorphism, without any definition of the particular polymorphisms claimed.

Finally, University of California v. Eli Lilly and Co., 43 USPQ2d 1398, 1404, 1405 held that:

To fulfill the written description requirement, a patent specification must describe an invention and do so in sufficient detail that one skilled in the art can clearly conclude that "the inventor invented the claimed invention." Lockwood v. American Airlines, Inc., 107 F.3d 1565, 1572, 41 USPQ2d 1961, 1966 (1997); In re Gosteli, 872 F.2d 1008, 1012, 10 USPQ2d 1614, 1618 (Fed. Cir. 1989) (" [T]he description must clearly allow persons of ordinary skill in the art to recognize that [the inventor] invented what is claimed."). Thus, an applicant complies with the written description requirement "by describing the invention, with all its claimed limitations, not that which makes it obvious," and by using "such descriptive means as words, structures, figures, diagrams, formulas, etc., that set forth the claimed invention." Lockwood, 107 F.3d at 1572, 41 USPQ2d at 1966.

An adequate written description of a DNA, such as the cDNA of the recombinant plasmids and microorganisms of the '525 patent, "requires a precise definition, such as by structure, formula, chemical name, or physical properties," not a mere wish or plan for obtaining the claimed chemical invention. Fiers v. Revel, 984 F.2d 1164, 1171, 25 USPQ2d 1601, 1606 (Fed. Cir. 1993). Accordingly, "an adequate written description of a DNA requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it; what is required is a description of the DNA itself." Id. at 1170, 25 USPQ2d at 1606.

Claim Rejections - 35 USC § 103

9. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

10. Claims 1-3, and 6-10 are rejected under 35 U.S.C. 103(a) as being unpatentable over rs5637 (dbSNP, ss7104, 1999) in view of Soderlund (US Patent 6,013,431).

Although the claims recite " a method for diagnosing a predisposition to fat deposition", all humans, to some degree, are subject to fat deposition. Accordingly, the intended use of diagnosis has been given no patentable weight because detection of either allele of a polymorphism is associated with predisposition to some degree of fat deposition. Claim 6 has been examined as dependent from claim 1 (see claim objection above).

The polymorphism denoted rs5637 in PLA2G1B, which is at position 7328 of SEQ ID NO: 1 has been determined. With regard to claim 3, ss7104 teaches that the polymorphism is an A or a G and provides sequences flanking the polymorphism. Although the disclosure in dbSNP (ss7104) does not specifically teach a method of detecting the variation, Soderlund teaches a method of detecting nucleotide variations using a method of obtaining nucleic acid from a subject, hybridizing an oligonucleotide complementary to a known sequence which is adjacent to the polymorphic variation, extending the oligonucleotide, and detecting the presence or absence of the polymorphic variation (see Figures 1-3). Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to use the method of nucleotide variation detection of Soderlund to detect the polymorphism denoted rs5637 in a subject for the purpose of characterizing the polymorphism. In detecting the presence or absence of the polymorphism with the method of Soderlund, the ordinary artisan would have been motivated to construct sequences adjacent to the polymorphic variation as taught by Soderlund, including the sequence of SEQ ID NO: 38, which is immediately adjacent to the polymorphism taught by rs5637. The assay summary teaches the nucleotide sequence flanking the polymorphic variation. Therefor, with the teachings of Soderlund, constructing a primer for extension analysis of the polymorphism would have been routine experimentation as Soderlund

specifically provides guidance in the selection of sequences for polymorphic nucleotide detection.

11. Claims 1-2, 4, 5, and 7-10 are rejected under 35 U.S.C. 103(a) as being unpatentable over rs1179387 (dbSNP, ss1634336, October 2000) in view of Soderlund (US Patent 6,013,431).

Although the claims recite " a method for diagnosing a predisposition to fat deposition", all humans, to some degree, are subject to fat deposition. Accordingly, the intended use of diagnosis has been given no patentable weight because detection of either allele of a polymorphism is associated with predisposition to some degree of fat deposition. Claim 4 has been examined as dependent from claim 1 (see claim objection above).

The polymorphism denoted rs1179387 in PLA2G1B, which is at position 9182 of SEQ ID NO: 1 has been determined. With regard to claim 5, ss1634336 teaches that the polymorphism is an C or an A (complement: G or T) and provides sequences flanking the polymorphism. Although the disclosure in dbSNP (ss1634336) does not specifically teach a method of detecting the variant, Soderlund teaches a method of detecting nucleotide variations using a method of obtaining nucleic acid from a subject, hybridizing an oligonucleotide complementary to a known sequence which is adjacent to the polymorphic variation, extending the oligonucleotide, and detecting the presence or absence of the polymorphic variation (see Figures 1-3). Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to use the method of nucleotide variation detection of Soderlund to detect the polymorphism denoted rs1179387 in a human subject for the purpose of characterizing the polymorphism. In detecting the presence or absence of the polymorphism

with the method of Soderlund, the ordinary artisan would have been motivated to construct sequences adjacent to the polymorphic variation as taught by Soderlund, including the sequence of SEQ ID NO: 40, which is immediately adjacent to the polymorphism taught by rs1179387. The assay summary teaches the nucleotide sequence flanking the polymorphic variation. Therefor, with the teachings of Soderlund, constructing a primer for extension analysis of the polymorphism would have been routine experimentation as Soderlund specifically provides guidance in the selection of sequences for polymorphic nucleotide detection.

Conclusion

12. No claims are allowed.

13. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Sitton whose telephone number is (571) 272-0752. The examiner can normally be reached Monday-Thursday from 8:00 AM to 5:00 PM and on alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla, can be reached on (571) 272-0735. The fax phone number for this Group is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public.

For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

Jehanne Sitton
Primary Examiner
Art Unit 1634
6/22/06